

T H E S I S

GENERAL PARALYSIS OF THE INSANE

WITH SPECIAL REFERENCE TO ITS  
AETIOLOGY, SYMPTOMATOLOGY, & MORBID ANATOMY.

COMPOSED BY

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## GENERAL PARALYSIS OF THE INSANE.

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Synonyms.—General Paralysis: Progressive Paralysis of the Insane: Progressive General Paralysis: General Paresis: Paralytic Dementia: Paretic Dementia.

This disease, with its obscure ætiology, its often remarkable and complex symptomatology, its progressive course, & its fatal termination, cannot but have a strong fascination for all engaged in the study of neurology. Probably no other nervous disease has had more attention paid to it by modern observers, from both its pathological and clinical aspects, and more especially from the point of view of morbid anatomy — naked eye and microscopical. In spite of this fact, however, its exact cause and nature are still far from being elucidated and remain a very fertile source of controversy.

I propose in this Thesis to give a short summary of the leading facts about the disease in the light of some of the latest researches on the subject, giving particular prominence to a few special points which are barely mentioned in the ordinary text-books, and which appear deeply interesting and important, such as the relation of the disease to Syphilis, etc.

HISTORY. The first known records of General Paralysis are by Willis in 1672, and Haslam in 1798. Before the first of these dates, nothing appears to have been known of the disease as a separate affection, and the

two writers mentioned had a very vague and incomplete knowledge of the subject, making reference to merely one or two of its distinctive features.

The first descriptions approaching completeness were given by Bayle (1) in 1822, 1825 & 1826, and by Calmeil in 1826 (2).

As late as 1828 Burrows (3) gave an account of a "chronic-muscular Paralysis" - undoubtedly identical with General Paralysis. He did not recognise the mental symptoms as being a part of it, and stated that it came on at various periods in the course of insanity. He also confused it with other Paralyses, such as hemiplegia.

In 1835 Prichard (4) gave a more definite and clearer account, but stated that the disease might commonly come on years after the commencement of an attack of insanity, and also that the physical features of the developed disease might co-exist with perfectly sound mental faculties. In fact all the older writers regarded General Paralysis as a complication or as one of the terminations of ordinary insanity.

Conolly (5) lecturing in 1846, gave a very complete description of the disease, and stated that before reading Calmeil's work he had no knowledge of such symptoms, but had since recognized several cases.

Since then, General Paralysis has been made a special line of research by a large number of neurologists and alienists, amongst whom in more modern times, Mickle and Bevan Lewis stand out prominently.

## INCIDENCE.

Age.- General Paralysis is usually seen between the ages of 30 and 55, and very seldom except between 25 and 60.

In proportion to the population, the greatest incidence is in the decade 40-50, although the greatest absolute number of cases occur between 30 and 40.

Burman (6) in a statistical paper based on 341 Consecutive cases gives the life-period 35 to 45 as furnishing the most numerous cases.

Mickle (7) thinks that General Paralysis occurs now-a-days earlier in life than formerly, judging from the statistics of the older writers, such as Bayle (1) who saw only 4 cases under 30 out of 182, and Calmeil (2) who only saw 2 altogether. Certainly cases under 30 are common enough now, although an allowance must be made for defective diagnosis in the time of the writers mentioned.

Statistics from the English Lunacy Blue Books prove that the disease occurs in England earlier in women than in men, and Burman (6) confirms this result.

Apparently well authenticated cases of General Paralysis in children are not very uncommon.

Alzheimer (8) in 1894 collected notes of 40 cases occurring between 10 & 16, in which the symptoms and post-mortem appearances were very similar to cases occurring in adults. He also reported a case of his own in which the first symptoms occurred at the age of 8, and the autopsy shewed typical appearances.



Joulouse (9) collected 16 cases of juvenile General Paralysis in which 15 years was the average age of onset.

Burman (6) out of his total of 341 gave 28 as under 30 and 12 over 60.

SEX- General Paralysis is much less common in women than in men. The proportion in which the two sexes are affected appears to be about 4 to 1, founded on the English Lunacy Commissioners reports for eight consecutive years (10). Burman (6) also arrived at exactly this result. The proportion has been variously stated by different observers as being from 10 to 1, to  $2\frac{1}{2}$  to 1. In the 10 consecutive years from 1892 to 1901, I find there were 192 males admitted to the Newcastle City Asylum suffering from General Paralysis and 39 females, which gives a proportion of almost 5 to 1.

The cause of the difference in the numbers of the two sexes affected is probably the greater prevalence of intemperance in various directions amongst males, their greater liability to overstrain of mind and body, and the more exhausting effects of sexual excesses. Mickle (7) states that "the male brain is innately more liable to organic disease than the female". Davean thought that the reason was a prophylactic influence exerted by the process of menstruation (11) and Lunier (12) appeared to confirm this view by stating that the proportion of female sufferers became greater after the menopause. Mickle (7) however, points out that statistics in this country shew quite the

opposite to this statement.

In France and Germany, the proportion of female General Paralytics is much higher amongst the lower than in the upper social classes, (4-1 Public Asylums:  $9\frac{1}{2}$ -1 Private Asylums).

General Paralysis is also said to be increasing in women, but this result of statistical comparisons is partly at least due to imperfect knowledge and defective diagnosis in former times, since the disease as it occurs in the female is certainly very often vague and difficult to recognise.

#### Condition as to Marriage.-

According to English Statistics compiled from the Lunacy Commissioners' Reports (10) the proportion of married General Paralytics to single ones is almost exactly 4-1, and to widowed ones more than 2-1. This is for the sexes taken together, and the figures do not vary much taken separately. This overwhelming majority of married General Paralytics is due to the fact that the time of life at which the incidence is greatest is also the time at which the most people are in the married state. Chapman (13) excluded this fallacy by finding the ratio of the General Paralytics in the different marriage conditions and in the various decades to the total population of the same conditions and ages. His results were: 16.5 single, 15.3 married, and 15.4 widowed General Paralytics per 100,000 of the same age (50-60) and the same condition as to marriage. Thus it would seem that the married have no special liability to the disease.

Social Position and Occupation.- The disease is more frequent in the lower than in the upper classes. Occupation has also some influence. Thus the Military and Naval services were found by Mickle (7) to furnish most cases, and the miners in the Northern Counties are notoriously liable to General Paralysis. The frequency amongst soldiers is generally attributed to the hard life, shocks and privations of war, alcoholic and sexual excess, and venereal disease, and in miners some of the same influences are at work.

Geographical Distribution.- It is rare in hot climates but this comparative exemption does not extend to new comers. Where the population is composed of both whites and blacks, a greater proportion of the former are affected.

There are three times as many deaths from General Paralysis in the principal towns as in the smaller towns and rural districts, whereas the death rate from all causes is much less than twice as great.

Ireland has proportionally very few cases, many of the large asylums containing none at all. Mickle (7) thinks that the Celt is less liable to the disease than the Saxon, "just as he exhibits less of the sustained effort, and sustained and applied cerebral energy of the Saxon".

The North of Scotland is also comparatively free from General Paralysis. Probably however, little importance should be attached to these racial differences, as Irishmen appear to be quite as liable to the disease when they come to live in the cities. In the



Isle of Man not a single case was recorded during the 6 years 1884 to 1890 (14)

Burman (6) gave an interesting list of deaths from General Paralysis in different localities, varying from 34.5 per cent in Chester to  $1\frac{1}{2}\%$  in Suffolk. Wakefield gave 20%, and Cambridge 9%. The percentages are taken on the total deaths in the institutions (5).

Aetiology:-

We come now to the most uncertain and debatable portion of the subject: the Causation of General Paralysis. It may be said at once that almost every writer has a different account to give, at least as regards the relative importance of the commonly received causes.

Such are: Intemperance in Alcohol, Sexual excess, overstrain of mind & body & syphilis, with many others of minor import, such as cranial injury, sunstroke, lead-poisoning etc.,

In dealing with these, the importance of making a distinction between predisposing and ~~exciting~~ causes is not at all apparent, especially when one considers the amount of uncertainty that exists upon the part played by each of them taken separately. The question what relation has General Paralysis to Syphilis? - is one that may be conveniently considered first.

Ever since the first recognition of the disease, the relation of General Paralysis to Syphilis has been a very keenly debated point amongst neurologists and alienists, and on looking back on the literature of



the subject, one meets with the most remarkable differences of opinion and varied results of the compilation of statistics. Thus, to take two extremes, Savage (15) has stated that something like 90% of cases of General Paralysis are caused by Syphilis, and Bevan Lewis (16) thinks that Syphilis plays but an unimportant part in the causation.

Between these there is a host of intermediate opinions. In what proportion of cases can a distinct history be obtained or objective signs of the disease be found? On turning to statistics, one is impressed by the enormous differences between the figures of various observers.

Jespersen (17) was able to obtain definite histories of Syphilis in 77% of his cases. Mendel found a history of secondary Syphilis in more than 50 per cent of his patients. Rieger (18) by combining the observations of "10 different and trustworthy" investigators, found that 399 out of 1000 General Paralytics were Syphilitic, or nearly 40 per cent. Taking females alone, Jacobson (19) found that 65 per cent of 116 cases had had Syphilis. (15)

Alzheimer in his 40 cases of juvenile General Paralysis (8) found that 36 of them had hereditary Syphilis, and is of opinion that General Paralysis arises from the same cause at more advanced ages also. Morrell-Lavallée (20) and Bélières in 1889 (21) came to the conclusion that the number of cases in which Syphilis plays a part in the causation of General Paralysis increases directly in proportion to the

care taken in analysing the history of each patient.

Founded on these facts is the doctrine that General Paralysis is essentially a syphilitic disease, first suggested by Esmerch and Jessen, afterwards upheld by Steenberg, Kjellberg and Jespersen, and still supported by a few modern alienists, amongst whom Hirschl of Vienna has strongly advocated the view in a paper based upon 175 cases 56% of whom he found certainly syphilitic, and 25% more probably so (22). Apart from statistics he brings forward three arguments to support the syphilitic theory:-

1. The anatomical process is a diffuse interstitial cortical encephalitis, analogous to interstitial hepatitis.

2. Reflex pupillary rigidity occurs almost exclusively in General Paralysis, Tabes, and Syphilis.

3. Various observers have seen remissions produced by specific treatment.

Against this theory there is much strong evidence, and most modern authorities do not consider the proofs by any means powerful enough to warrant so sweeping a conclusion. Thus Mickle found that out of all the cases admitted into the Asylums of England and Wales during four consecutive years, only 1.1 per cent had Syphilis assigned as a cause, and allowing a sufficiently wide margin for inaccuracy this is a distinctly striking result.

Burman in 341 cases (6) found Syphilis given as a cause in one case. The experience in the Newcastle City Asylum confirms these results, and although a reasonable amount of care is taken to ascertain the previous

histories of the patients, the number of cases in which an account of Syphilis can be obtained, or in which any objective signs of the disease can be detected, is remarkably small. Thus there were 80 cases of General Paralysis admitted during the years 1899, 1900 and 1901, and in not a single case is Syphilis traceable.

Readily granting that complete and accurate histories could not be obtained in all the cases, and making full allowance for other sources of fallacy, such as the well known reluctance of patients and their friends to confess to the syphilitic infection, still one is, I think, forced to the conclusion that at least some of the 80 cases mentioned were not in any way related to Syphilis.

Turning from statistics, one of the strongest arguments against the theory is the fact that definite Syphilitic changes are not found in the brain after death. Van Gieson (23) says that there are three forms of cerebral Syphilis.

1. The multiple variety, appearing in the form of plaques.
2. The gummatous form.
3. The form in which there are minuter lesions of the blood vessels.

Now the appearances in General Paralysis certainly correspond to none of these. The increase in the nuclei of the perivascular sheath is quite distinct from the definite endarteritis of Syphilis, and Hirschl's first argument given above appears to be an unjustified attempt to make the definite morbid



changes fit in with his clinical statistics.

Again the same writer's statement concerning remissions produced by anti-syphilitic treatment is by no means supported by the results obtained by the treatment in the Newcastle City Asylum. Experience here shews that the patient is either not appreciably affected by the treatment, or in a large number of cases is very prejudicially affected, even in some of the few cases with a distinct syphilitic history. In the face of these facts it is difficult to see how one can accept the syphilitic theory, and indeed there are several ways of accounting for the figures brought forward by its supporters:-

(a) Real manifestations of tertiary Syphilis undoubtedly often clinically resemble General Paralysis, and it is by no means impossible to mistake one for the other. I shall allude to this point below.

(b) Syphilis may be specially prevalent in the districts from which the statistics are drawn. The value of the figures would be greatly enhanced if the proportion of syphilitics to the general population were also given.

(c) As Clouston (24) points out, the syphilis may in many cases be the result of the reckless exposure to risks of infection, and the sexual excesses which often characterize the commencement or prodromal stage of the disease, especially in a district where Syphilis is prevalent.

(d) The Syphilis may have no connection and be purely accidental. Fournier (25) states that this is invariably the case when we are dealing with examples of true General Paralysis.



That mental disturbances, more or less severe, associated in some cases with local tremors & other physical symptoms, can be the definite result of Syphilis has been recognised for some time. Clifford Allbutt (26) in 1873 indicated this clearly, giving accounts of several cases in which such symptoms disappeared rapidly under treatment. Fournier (25) in 1879 stated definitely his belief in the existence of brain diseases due to Syphilis, resembling General Paralysis, but distinct from it. Clouston (26) describes a separate form - Syphilitic insanity, which differs from General Paralysis in its anatomical appearances, and notably in the prognostic characteristics, being amenable to specific treatment in a number of cases.

Savage (15) Kiernan (27) and others have stated that the syphilitic cases cannot be distinguished clinically from ordinary cases, but this assertion, even if true, does not disprove the existence of the two different conditions, since General Paralysis far from being a clinical unity, is an extremely protean malady.

Rejecting, then, as untenable the doctrine that General Paralysis is essentially a syphilitic manifestation, we come to the question, is there any relation between the two diseases? The majority of modern authorities especially in this Country reply in the affirmative, and the most reasonable view appears to be that Syphilis acts as an occasional predisposing cause, preparing the soil, weakening the organism, and rendering the brain more liable to the onset of the disease.

The exact part played by Heredity in the causation has also been disputed. One fact appears to be certain that it has not as much influence as in other forms of Insanity. Results obtained from the analysis of the Lunacy Blue Books shew that in about 15 per cent of cases of General Paralysis is Heredity stated as a predisposing cause, whereas 20 per cent of the total admissions are given as hereditary.

The statistics of many others prove the same thing.

In many cases a family history of other neuroses, such as Epilepsy, Alcoholism, etc., can be obtained, and further a General Paralytic often transmits a tendency to nervous disease, more frequently gross cerebral affections than insanity, to his offspring. "A life absorbed in ambitious projects, with all its strenuous mental efforts, its long sustained anxieties, deferred hopes & straining expectations, or any prolonged & violent, or sudden and frequent play of ill-regulated passions, frequently repeated outbursts of rage, smouldering flames of envy, jealousy or unrestrained sexual passion, the disintegrating influence of prolonged anxiety, of worry, of afflictions and losses, the similar influence of exaggerated selfishness and ambition, the concussion of moral shock, may both preface the way for and lead up to General Paralysis " (Mickle) (7).

Relation to previous Mental Disorder or Defect.

Previous attacks of insanity are not common in General Paralytics' histories, and many of those cases recorded are probably cases of long complete remission, which are not of uncommon occurrence. Köhler (28) stated that General Paralysis may supervene in Idiocy or Imbecility, & this statement has been supported by others. It also sometimes appears to originate in ordinary attacks of Insanity, but many errors have to be excluded in considering examples of this kind, which may be cases of General Paralysis with a long prodromal stage, or with slight or absent physical signs. On the other hand there is no reason to believe that such patients are specially exempt from the disease.

Mendel (29) thinks that the malady cannot come on in chronic insanity without some new factor leading to it, & Mickle (7) is also of this opinion.

Sexual Excess is commonly believed to be a fertile cause of General Paralysis, but here again opinions vary very widely. Many alienists have ascribed to it the most prominent part in the causation, & a few, such as Neumann (30) & Cavalier (31) went even further and stated it to be the exclusive cause. Most English authorities, such as Bevan Lewis (16), Clouston (24) Savage (15) Maudsley (32) Sankey (33), Shepherd (34) etc., certainly look upon this as a most important & frequent cause.

Mickle (7) thinks that though it acts occasionally, it is by no means the most fertile cause, and Mendel (29)



gave it a very insignificant place. Statistics gave  $2\frac{1}{3}$  per cent out of 4284 cases of General Paralysis where Sexual Excess was assigned as a cause, according to Mickle (7) Burman (6) found 5 cases out of 341, a still smaller percentage.

The mere fact that Sexual Excess can play no part in the causation of Juvenile General Paralysis negatives the supposition that it is the exclusive cause. It must also be borne in mind that Sexual Excess is a frequent early or prodromic symptom, & again Wilson (35) would have us believe that it is simply a result of the full blooded temperament & sensuality characteristic of the diathesis.

It is generally agreed that Masturbation rarely leads to General Paralysis, & this decidedly weakens the argument for sexual excess, as probably the former vice is more exhausting than the latter.

It is probable that sexual excess acts, not strictly speaking as an exciting cause, but by exhausting & weakening the nervous system, & more especially when combined with Intemperance in Alcohol & overwork. Mickle (7) also points out that the age at which sexual excess is most frequent, by no means corresponds to that during which most General Paralysis occurs.

#### Intemperance in Alcohol:-

Is undoubtedly an important factor. Most statistics point to it as being the most frequent, and at the Newcastle City Asylum a history of Alcoholism is very frequently obtained, especially amongst men. It, however, is also far from being a universal cause, &



I have myself seen several cases in which it could with reasonable certainty be excluded. It probably acts most frequently in combination with other causes.

Most observers bear testimony to the influence exerted by Overwork of body or brain, especially when accompanied by long continued worry & anxiety, & still more so when the strength is sustained by Alcohol. Christian (36) from his wide experience of General Paralysis summarizes his conviction as to its causation as follows:-

1. Predisposing Causes: Comparative weakness of brain congenital or acquired in the majority of cases.
2. Exciting causes: Anything which causes prolonged fatigue of the brain (trouble, insomnia, excess of study or physical labour) The latter causes may be comprised in one word "overwork".

With regard to the manner in which this acts he says "if we examine the causes which I have enumerated, those whose action is sudden & violent (fear etc) as well as those which only act slowly & insidiously (trouble etc) it cannot be doubted that they cause changes, rapid or slow, in the cerebral circulation.

#### Other Causes

Several instances have been recorded of General Paralysis arising from Sunstroke & Cranial Injury, in some cases the symptoms coming on very rapidly after the injury.

Is supposed by the Germans to be also a cause.

#### Summary of Causation.

From what has been said on each of the commonly received causes, it will be seen that the greatest uncertainty & doubt prevails on the subject. It seems clear that no one of the influences mentioned can be a universal cause, and the question then arises - Is General Paralysis caused sometimes by one, sometimes by another, and sometimes by a combination of 2 or more of these Agencies, or is there some one as yet undiscovered exciting cause at the bottom of every case? In the present state of our knowledge the first of these views is the more probable.

#### Symptoms.

I do not propose under this heading to give the well known classical symptoms & description of the course of this disease, with its insidious commencement, its delusions of grandeur & progressive dementia, its facial tremors, unequal pupils etc. All of this is sufficiently well known and described by the text books. There are, however, a few points in connection with the symptomatology, which are worth just glancing at. First with regard to Mental State:- The possibility of having all the physical features of the developed disease, combined with a thoroughly sound mental condition, has been and is still asserted, chiefly by General Physicians, who certainly if such cases do exist, would have a greater chance of seeing them than

the mental specialist, but the pitfalls of error are numerous. In the first place, it is often a difficult matter to exclude a slight amount of Dementia in a patient, especially for a general physician not experienced in examining the mental condition. Again, it is not always easy to elicit delusions of wealth or grandeur, especially in women, and the statements of friends are by no means trustworthy. In the case of a female patient at present under my care, there are characteristic and well marked changes, lip tremors, exaggerated knee jerks, with simply some slight scarcely noticeable excess of bien être at times & emotional irritability; she has been in this condition for some time; yet I have seen her maniacal, & she has expressed the most absurd delusions.

In females, the mental condition varies much more than in males. During my experience of nearly  $4\frac{1}{2}$  years in the Newcastle City Asylum, I have only seen 2 cases in which marked grandiose delusions were present, and in both these instances, there were periods of intense depression and hopelessness. There is often some optimism exhibited, without definitely expressed delusions. In one case of a Melancholic type, the patient could never be got to exaggerate her own possessions or powers, but would enlogize her husband (who was a very ordinary man) in the most extravagant style - Simple gradually increasing Dementia, without marked optimism or depression is common.

Hallucinations of Sight are seen, but they are certainly rare. In 2 cases which I have at present under my care, the patients describe vividly the appearance of



imaginary relatives standing by the bedside and address them, while one stated that she had seen "Christ in the room at night". Melancholia is commoner amongst women than men.

A peculiar form of hallucination has been described, termed Verbal-Motor Hallucinations, in which the patient imagined that someone was speaking to her, but denied hearing any sounds. She could understand the words, but could not hear them.

Physical Signs:-

The most constant & important are the ocular anomalies, and of these, curiously enough, the oldest and most commonly known and described, namely irregularity of the pupils, is probably of least importance when taken by itself, since it is so very often present in health, & in other slight nervous affections. The reactions should always be tested when inequality is present.

Bevan Lewis has lately (16) published a method of classification of varieties based upon the oculomotor anomalies. He found, after very careful analysis of 147 cases, that they fell into 5 groups with distinct pupillary changes, associated with more or less definite and distinctive bulbar, spinal & mental changes, as follows:-

Group 1.

- a. Paralytic Mydriasis: a partial reflex iridoplegia (light)
- b. Increased myotatic irritability.
- c. Excessive Facial tremor & Speech troubles.



Group II.

- a. Mydriasis with associative iridoplegia, rapidly passing into the cycloplegic form - an early symptom.
- b. Frequent myotatic excess, but no contractures.
- c. Late speech troubles.
- d. Acute excitement with frequent convulsions.
- e. Very rapidly fatal course (Preponderance of Syphilitic history)

Group III.

- a. Spastic Myosis: Complete reflex iridoplegia
- b. Absent or greatly impaired knee jerk.
- c. Failure of equilibration: locomotor ataxy, defective sensibility.
- d. Very defective articulation.
- e. Much optimism & excitement.

Group IV.

- a. Late eye symptoms: paralytic mydriasis. A partial reflex iridoplegia (for light only).
- b. Ataxic paraplegia (lower extremities only)
- c. Great facial ataxy with extreme troubles of speech.
- d. Epileptiform seizures ushering in pronounced mental enfeeblement.

Group V.

- a. No oculo-motor symptoms, beyond occasional inequality.
- b. No contractures, but notable myotatic excess.
- c. No disturbance of equilibration, locomotion or sensation.

d. Speech troubles not pronounced.

e. Epileptiform seizures very rare, but from the first progressive deepening Dementia.

This system seems likely to be of real practical use, and is a distinct step towards a more definite comprehension of the various forms of the disease, which without such a scheme are chaotic in the extreme. When a case is thus classified, it is now possible to form to a certain extent what the course of the disease will be, whether rapid or slow, complicated with many seizures or comparatively free. It is true that a classification into 5 groups was given by Mickle (7) years ago, based mainly on the pathological changes found after death, and especially the amount & distribution of decortication combined with some clinical (notably mental) features, but although as he pointed out, a system of classification to be enduring & useful, must have a pathological basis, his own adaptation of this principal has not been generally adopted, mainly I should think, because his distinguishing features were too vague and ill defined.

Lewis also points out that cases, in which there is early loss of light reactions & gradual failure of accomodation (cycloplegia) combined with mydriasis, are often associated with a Syphilitic history.

The External Ocular Muscles are rarely affected, usually only temporarily, & as shewn by Mickle often in cases where tabetic signs are present (3rd Group) thus in the case of a woman with absent knee jerks,

myosis, & almost complete reflex iridoplegia there was temporary ptosis & squint lasting over two months.

Clifford Allbutt (26) shewed in 1868 that Atrophy of the Optic Disc could be detected by the ophthalmoscope in a very large percentage of cases; this is not a very early symptom.

Leaving the eyes we turn to the Spinal Reflexes, & this lands us in the midst of another of the battle grounds of neurologists so common in connection with General Paralysis, viz:- What relation has tabetic General Paralysis to Tabes Dorsalis?

It is stated by some writers that General Paralysis commonly supervenes in Tabes, and thus forms a complication. They explain this by supposing that the cerebral derangements are due to ascending changes produced by direct continuity of tissue from the cord to the cortex. Others say that true Tabes very rarely becomes General Paralysis, and that the symptoms in ordinary cases are merely those of a tabetiform state. Lewis's view is that these primary symptoms of cord implications are due to a genuine angio-neurosis, which may ultimately lead to true degenerative changes in the posterior columns. This process he regards as secondary to the destructive irritative lesions of the cord. There are good reasons for this view:-

1. In some of the most marked cases of tabetic General Paralysis, no alteration is found in nerve fibres of the posterior columns, while in true Tabes there is always destruction of the fibres.

2. There is an increase of size in vessels of posterior

columns.

3. In a large number of the tabetic cases, the knee jerks return, the tabetic gait disappears, and there is full development of the cerebral symptoms.

With regard to Facial Tremors - the brow muscles are comparatively rarely involved compared to the lip muscles, the former are more affected in Alcoholism.

The Tongue is almost always very tremulous, and can seldom be protruded straight, but there is at present a male patient in this Asylum with extreme impairment of speech and marked lip tremors, whose tongue protrudes straight and is kept perfectly steady. In a number of cases the convulsive seizures affect the face only.

#### Morbid Anatomy.

The changes found in General Paralysis are fairly constant & well defined. To take them briefly in order as I have observed them in the post mortems, during 4 consecutive years at the Newcastle-on-Tyne Asylum, and more especially in 47 brains of General Paralysis, examined by myself -

The Skull Cap is sometimes thickened, but this is by no means constant; it is occasionally thinner than normal.

Membranes:- The Dura Mater is often thickened, but only rarely unduly adherent to the skull cap, base of skull or leptomeninges. In a large percentage of cases "Pachymeningitis haemorrhagica interna" or "Arachnoid



Cyst" is seen, often covering one or both hemispheres, more or less completely, and in various stages of colour, from a dark red to a dirty whitish-yellow. With regard to the exact pathology of this appearance Goodall (37) has recently produced a very similar condition, when experimenting on rabbits, and states that in no case was there an inflammatory exudate, or even an excess of white corpuscle in the early stages, a fact which materially supports the purely haemorrhagic theory of origin. The Pia Arachnoid is thickened in all cases. In the majority of cases there is some localization of the thickening, usually to the Frontal & Parietal regions, and in & about the Sylvian Fissure. In some cases, the thickening is general, but in most, the occipital region escapes more or less completely. In some cases the membranes are very thick, spongy & soft (in 2 cases I examined, nearly  $\frac{1}{4}$  inch in places) with much fluid in the meshes, and in others dense in texture & very tough.

Out of the 47 postmortems mentioned above, I found adhesion of the membranes absent in only 3 instances. The adhesion is most marked in the Frontal, Parietal & Supra Marginal gyri and occasionally the upper part of the Temporo-sphenoidal; the tip of the Occipital is nearly always free.

Opacity of the membranes I have recorded as being marked in 24 cases & slight in 23.

The Convulsions are usually described as being flattened & shrunken, & although this is often more marked on one side than the other, taken altogether there is no great preponderance on either side.

With regard to locality the parts most affected are the profrontal & Rolandic Areas; the cortex is very usually atrophied in these regions. In colour & consistency it varies, sometimes dense and sometimes softer than usual. Localized softenings of the cortex are uncommon. The Lateral Ventricles are almost always dilated with granular ependyma, a change also found in the 4th Ventricle, and the amount of fluid varied from 2 to 12 oz.

Other notable changes are not common.

Outside the brain the only organs commonly affected are the kidneys, which are very often granular & contracted.

#### Microscopical:-

Fresh sections from the motor area stained with A.B.B. by Bevan Lewis' method (15) shewed in most cases, a great number of spider cells, destruction & degeneration of the proper nerve cells, proliferation of nuclei in the perivascular sheath, and general dark staining of the neuroglia. The Pia was thickened, and in the case of the marked adhesions mentioned above sections through the Pia with some of the retained cortical matter, shewed a most extraordinary development of spider cells, the fibres of which were continuous with the Pia. Many of the nerve cells were undergoing pigmentary degeneration. In the Cord, one sometimes finds degeneration of the posterior columns or of the lateral tracts.

Opinions as to the nature of General Paralysis vary and have varied between wide extremes.

The main points at issue are:-

a. Whether cerebral hyperaemia is of primary importance or produced as a secondary phenomenon.

b. Whether the essential pathological changes are of an inflammatory or of a degenerative nature.

c. Whether the change if inflammatory is interstitial or parenchymatous.

Rokitansky (38) attributed the change primarily to the connective tissue, partly due to hyperaemia, and partly to an interstitial inflammation, this resulted in disorganization of the Cortex, the nerve cells and fibres being altered and broken up. Mendel (29) regards it as a diffuse interstitial cortical encephalitis resulting in brain atrophy, in which the nerve cells and membranes are secondarily affected. Hirschl of Vienna (22) and many others support this latter view.

Bevan Lewis (16) looks upon the change as commencing as an inflammatory lesion of the vessels of the Pia, which spreads downwards to the deepest layers of the cortex, and the nerve cell degeneration as being Secondary. He states that the normal lymph channels round the vessel being disorganized, the spider cells take upon themselves the functions of scavengers and remove effete matters, while they also have a disintegrative effect on the nerve cells, and cause their destruction.

The evidences of inflammatory action are, according to



Mendel & others, the hyperaemia of membranes, cell multiplication, development of spider cells, and increased excretion of intercellular substance. Some have quoted the temperature which is often raised, as being confirmatory evidence of inflammation.

We have thus already 2 sets of opinions, both upholding inflammatory action - in the one, the interstitial tissue being the primary seat, & in the other, the vessels. Meschede (39) regarded the commencement of the process to be parenchymatous inflammation, principally affecting the ganglionic nerve cells. Clouston (24) and Weigert (40) regard the change as being primarily parenchymatous with resulting secondary vascular and neuroglia changes. Clouston (24) thinks the nerve cells undergo degeneration in the first place - he says "the degenerative theory is based on the whole etiological, clinical and Pathological history of the disease, while the inflammation theory almost exclusively on visible pathological changes in the vascular or lymphatic elements of the cortex".

He looks upon the disease as being equivalent to a premature senile condition " Senility being the slow physiological process and General Paralysis being the quick pathological one. He explains the elevation in temperature as being due to a disturbance of the heat regulating mechanism in the cortex. Mickle also thought the change was fundamentally parenchymatous when the second edition of his work was published in 1886, but he has since modified his views, and in 1894 (7) he expresses his opinion, that some of the

clinical varieties were due to the variations in the essential lesions, whether primarily affecting one set of tissues or another.

In conclusion I shall refer very briefly to two other points of clinical interest - viz: the Prognosis and the Duration.

### Prognosis.

This would seem to be absolutely & without exception unfavourable. Cases of "recovery" have certainly been recorded, but according to Clouston (24) & Mickle (7), these may be regarded of 2 kinds:-

1. Long continued remissions, certain sooner or later to relapse.
2. Pseudo. General Paralysis, principally syphilitic.

### Duration

This would certainly appear to vary with the locality. At the Newcastle City Asylum, the average duration was calculated at 20 months for males, and 24 for females. At Devon County Asylum, 17 months for both sexes.

Cases lasting 16 to 20 years have been recorded, and also cases lasting 6 months. The duration is distinctly longer in women than in men, and in the upper classes than in the lower.

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AUTHORITIES QUOTED IN COMPOSITION OF THESIS ON  
GENERAL PARALYSIS OF THE INSANE.

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- |  |                      |
|--|----------------------|
| 1. Bayle.                                    | 26. Clifford Allbutt |
| 2. Calmeil.                                  | 27. Kiernan.         |
| 3. Burrows.                                  | 28. Köhler.          |
| 4. Prichard.                                 | 29. Mendel.          |
| 5. Conolly.                                  | 30. Neumann.         |
| 6. Burman.                                   | 31. Cavalier.        |
| 7. Mickle.                                   | 32. Maudsley.        |
| 8. Alzheimer.                                | 33. Sankey           |
| 9. Joulouse.                                 | 34. Shepherd         |
| 10. English Lunacy Commissioners.            | 35. Wilson           |
| 11. Davean.                                  | 36. Christian.       |
| 12. Lunier.                                  | 37. Goodall.         |
| 13. Chapman.                                 | 38. Rokitansky.      |
| 14. Isle of Man Reports<br>from 1884 to 1890 | 39. Meschede.        |
| 15. Savage.                                  | 40. Weigert.         |
| 16. Bevan Lewis.                             |                      |
| 17. Jespersen.                               |                      |
| 18. Rieger.                                  |                      |
| 19. Jacobson                                 |                      |
| 20. Morrell-Lavallée                         |                      |
| 21. Bélières.                                |                      |
| 22. Hirschl.                                 |                      |
| 23. Van Gieson.                              |                      |
| 24. Clouston.                                |                      |
| 25. Fournier.                                |                      |